# Utah Diabetes Practice Recommendations 2004

Diabetes Management for Adults

# Table of Contents




This publication was supported by Cooperative Agreement Number U32/CCU822702-01 from the Centers for Disease Control and Prevention. Its contents are solely the responsibility of the Utah DPCP and do not necessarily represent the official views of the Centers for Disease Control and Prevention

## Endorsements

The following professional associations and groups have reviewed the sections of the Utah Diabetes Practice Recommendations that apply to their respective clinical areas of interest. They have endorsed these Recommendations, to the extent they apply to their clinical areas, and found them to be consistent with applicable standards of care for men and women with diabetes. In extending their endorsement, it is recognized that these recommendations, while outlining a general course of action for the majority of patients, do not substitute for informed clinical judgment on the exact course of treatment for individual patients.

Association of Diabetes Educators of Utah

Utah Academy of Family Practice

Utah Academy of Physician Assistants

Utah Chapter, American College of Physicians

Utah Department of Health

Utah Dietetic Association

Utah Diabetes Prevention and Control Program Advisory Board

Utah Nurses Association

Utah Nurse Practitioners

Utah Ophthalmology Society

Utah Optometric Association

Utah Pharmaceutical Association

Utah Podiatric Medical Association

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Special thanks to Betsi Briones for the charts and many revisions in these recommendations

# UTAH DIABETES PRACTICE RECOMMENDATIONS 2004

## Introduction

The Utah Diabetes Prevention and Control Program (DPCP) recognizes the importance of optimizing care for patients with diabetes. In promoting this objective, the DPCP organized a committee of interested health care professionals to develop the **Utah Diabetes Practice Recommendations - 2004** (UDPR). The recommendations are intended to foster current diabetes care practices, to provide useful outlines to guide healthcare professionals in screening and diagnosing people with diabetes, and to promote appropriate diabetes management. The materials in the UDPR build upon and complement national and regional diabetes protocols. Members of the UDPR Committee have identified decision points to assist the clinician in providing consistent and appropriate diabetes care for their patients.

Well designed and effectively carried out studies such as the **Diabetes Control and Complications Trial (DCCT)** and the **United Kingdom Prospective Diabetes Study (UKPDS)** have demonstrated convincingly that blood glucose control significantly affects the development of complications in individuals with either type 1 or type 2 diabetes. A direct link between blood glucose levels and the risk for complications has been firmly established, despite the fact that other factors such as genetics also play a significant role.

Providers should encourage individuals with diabetes to aim for the lowest blood glucose levels that do not place them at undue risk for hypoglycemia. The studies also show that any improvement in glucose control has the effect of slowing both the development and the progression of microvascular complications. Data from the UKPDS have shown the linear relationship between glycemic levels and the risk for complications. For each percentage point decrease in A1c, there was a 35% reduction in the risk for microvascular complications.

Clinical judgment is necessary to identify those patients for whom near normal glycemic control might not be appropriate. Near normalization of glycemic levels requires active participation by the patient and is not advisable for those who are neither capable nor willing to actively participate in their own diabetes management. It is not advised for very young children and may not be indicated for those whose impairments may compromise their ability to fully appreciate the regimens set forth in this document. There are some data that indicate that hypoglycemia in children could cause impaired brain development before the age of seven, while in older patients, hypoglycemia may lead to stroke or heart attack.

## Acknowledgement

The DPCP acknowledges the fine work and efforts of the Diabetes Management Team and the Primary Care Clinical Program at Intermountain Health Care (IHC) in the development of the UDPR. Much of the material incorporated in the UDPR closely follows the Care Process Model outlined in the 2003 update of IHC's *Management of Adult Diabetes,* and is used with permission kindly provided by IHC.

# Summary of Key Treatment Targets

| Measure/Test | Target | Comment | Frequency |
|---|---|---|---|
| **HbA1c** | **<7.0%** | As low as possible without significant hypoglycemia | Test at least semi-annually |
| **Blood Pressure** | **<130/80 mm Hg** | <125/75 for patients with nephropathy | Check at each office visit |
| **LDL Cholesterol** | **<70-100 mg/dL (depending on presence of CVD)** | Data suggest treatment with statins may be appropriate for people age >40 with total cholesterol ≥135 mg/dL | Test at least annually |
| **Urine Microalbumin or Microalbumin/ Creatinine Ratio** | **<30 mg per 24 hours or <30 mg/g of creatinine** | Use one of the following:<br>Micral (dipstick)<br>Spot urine<br>24 hour urine<br>Timed urine (<20 mcg/min) | Test at least annually |
| **Dilated Eye Exam** | **Normal** | High risk should be tested more frequently; low risk may require less often | Check annually |
| **Foot Exam** | **Identify Level of Risk** | Check every visit if significant vascular disease, poor protective sensation is present, or if identified as high risk | Complete an annual evaluation of pulses, test with monofilament fiber for loss of protective sensation, and question carefully about claudication |

# Guidelines for Frequency of Lab Tests and Examinations

| | |
|---|---|
| **Examination** | Every 3 months for those who are not meeting blood glucose or blood pressure goals, on new therapy, on intensive insulin therapy, or with evidence of progression of microvascular or macrovascular disease<br><br>Every 6 months for those who are meeting blood glucose and blood pressure goals, are not on new therapy, and do not have evidence of progression of microvascular or macrovascular disease |
| **Hemoglobin A1c** | Same as for examination above |
| **Blood Glucose** | If patient is self-monitoring blood glucose and records are acceptable: Optional<br><br>If patient is not self-monitoring blood glucose: Test when fasting at each examination visit and correlate with A1c |
| **Blood Pressure** [1, 2] | Check and record at every visit |
| **Foot Exams** [3] | 1. Screen feet annually: physical exam and sensory exam using a monofilament<br>2. Categorize findings: low or high risk<br>Low risk: none of the 5 high risk characteristics listed below:<br>High risk: one or more of the following:<br>Loss of protective sensation<br>Absent pedal pulses<br>Severe foot deformity<br>History of foot ulcer<br>Prior amputation<br>3. High risk: screen at every visit |
| **Dilated Eye Exam** [4] | Annually for most patients with mild or no NDPR or microaneurisms, biennially for patients in good control with advise from an eye care professional |
| **Microalbumin** [5,6,7] | Annually |
| **Fasting Lipid Profile** | Annually |
| **Influenza Vaccine** | Annually |
| **Pneumococcal Vaccine** | Once before age 65. Consult physician about revaccination after 65 |
| **Self-Management Education** | 1. Upon diagnosis<br>2. When there are significant changes in therapy; the patient is not meeting targets; for pre-pregnancy counseling; and newly diagnosed gestational diabetes<br>3. Annually reassess need for education |
| **Refer to Specialists** | 1. As needed, when not meeting targets<br>2. As needed, when complications are noted |
| **Dental Exam** | At least annually for preventive care |

1. If BP is 130-139/80-89 initiate exercise and nutritional intervention, if not effective use ACE-I, ARB or Thiazide diuretic; if BP >140/90 initiate lifestyle modification + ACE-I, ARB or diuretic; if >150/90, consider initial two drug therapy with ACE-I or ARB + Thiazide diuretic
2. Unless contraindicated, use low dose aspirin as a prophylactic measure at onset of vascular risk and/or after age 40: (Low dose: 81 mg to 325 mg every day)
3. Refer to "Feet Can Last a Lifetime" packet for additional foot screening information (www.ndep.nih.gov).
4. Exception: Examine when planning pregnancy if possible and in first trimester with close follow-up
5. Screen for protein before testing for microalbumin. If protein is present, it is not necessary to perform any tests for microalbumin.
6. Consider using ACE inhibitors if microalbumin levels are >30mg/24 hours as determined by a 24 hour urine collection or spot urine microalbumin/creatinine ratio >30 (on at lease two separate occasions).
7. Exception: Screen in first trimester in pregnancy.

# Diabetes in Utah and the United States

### Prevalence

Diabetes has received considerable attention in the health care community and the news media in recent years due to the rapid increase in prevalence rate. It represents a growing public health and clinical concern in Utah and the United States (U.S.). The most recent estimate for diabetes prevalence in the U.S. shows 18.2 million people have diabetes. Unfortunately, over 5 million of those with diabetes are unaware they have the disease. The Utah Health Status Survey indicates that there were 120,000 Utahns with diabetes in 2001, a 48% increase over the 81,000 in the same survey in 1991. Of those Utahns with diabetes, an estimated 40,000 are untreated because they have not been diagnosed.

### Complications

Diabetes is responsible for or associated with a number of serious and potentially fatal complications. It is estimated that up to **75% of individuals with diabetes die prematurely from heart disease.** Diabetes increases the risk of **heart attack and stroke** two to four fold; it is responsible for about half of all new cases of **kidney dialysis,** and is the leading **cause of blindness** among working age adults. Over half of all non-traumatic **lower extremity amputations** are related to complications of diabetes.

### Costs

Diabetes is a common and potentially disabling, chronic disease that costs this country over $132 billion a year. Studies indicate that Medicare costs for people with diabetes are double the costs for those without diabetes. In the Medicaid population and among the uninsured, the cost ratios are 4:1. In most other groups costs are triple those of people without the disease.

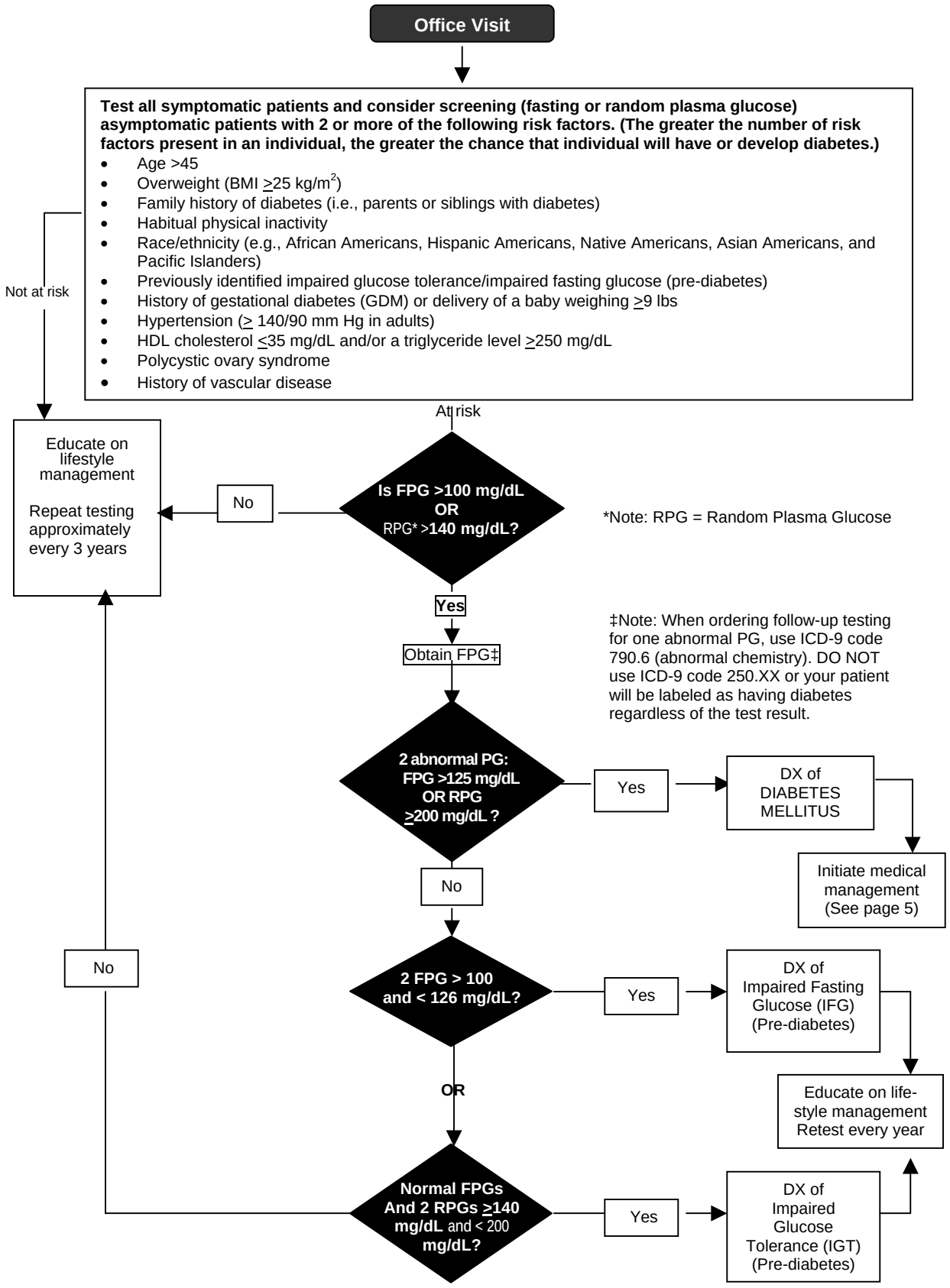
### Pre-diabetes and metabolic syndrome

Diabetic risk factors of *impaired glucose tolerance* (IGT) and *impaired fasting glucose* (IFG) have come to be known as pre-diabetes. The American Diabetes Association estimates that 22 million people in the U.S. have this condition that puts them at high risk for diabetes. Many people with pre-diabetes also fit into a category known as *metabolic syndrome*. A patient can be considered to have *metabolic syndrome* if he or she has any 3 of the following 5 criteria established by the third Adult Treatment Panel (ATP III) of the National Cholesterol Education Program as indicated below:

- Increased waist circumference (>40 inches in men; >35 inches in women)
- Plasma triglycerides $\geq$150 mg/dL
- Plasma high-density lipoprotein (HDL) cholesterol <40 mg/dL (men) or <50 mg/dL (women)
- Blood pressure $\geq$130/85 mm Hg
- Fasting plasma glucose $\geq$100 mg/dL

**Patients with either metabolic syndrome or pre-diabetes should be regularly screened for diabetes mellitus.**

## DIABETES SCREENING PROTOCOL

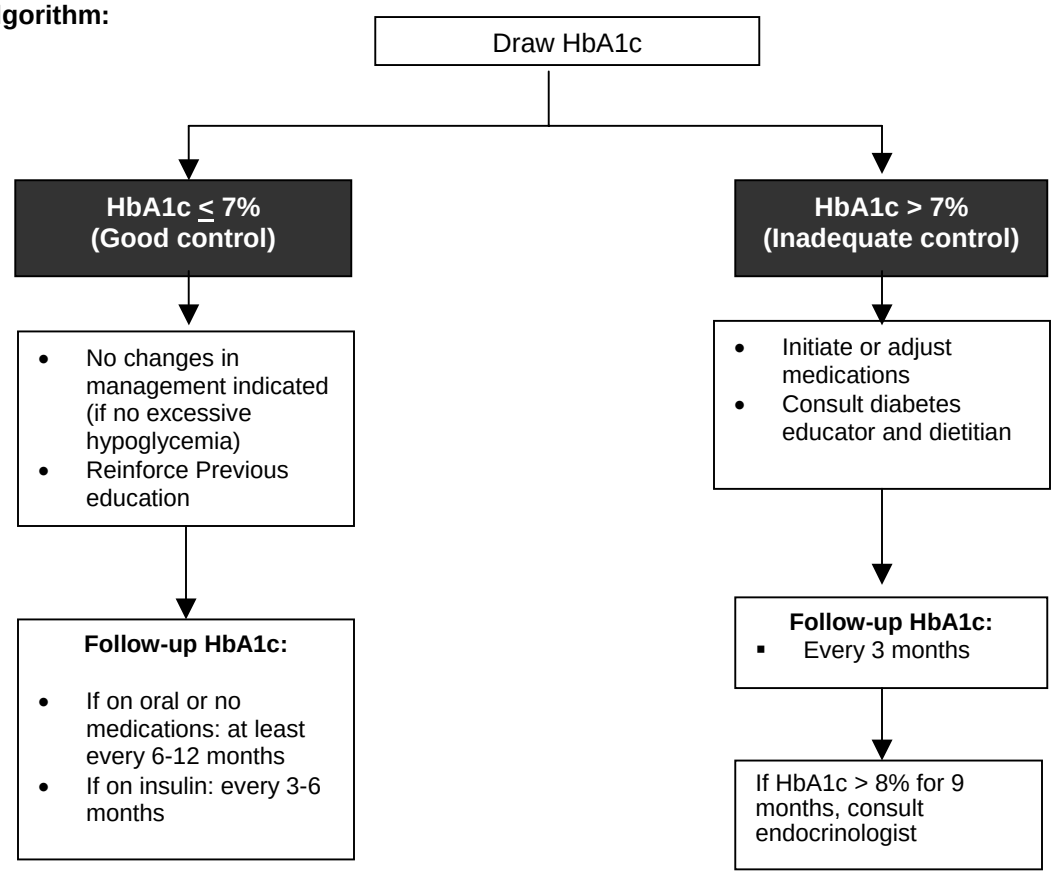


## THE ROLE OF HbA1c IN DIABETES MANAGEMENT

HbA1c testing is *not* recommended as an initial screening test for diabetes mellitus, nor does it dictate day-to-day management of diabetes. Rather, it is an indication of the overall trend of blood glucose levels for the previous three months. Monitoring of glycemic status is considered a cornerstone of diabetes care and affects how physicians and patients adjust medical therapy as well as behavioral therapy (e.g. diet and exercise). The better the diabetes control, the lower the HbA1c, and the fewer the complications. The HbA1c value can also be used to validate (or call into question) the patient's home record of blood glucose readings and/or random FPG testing performed in the office.

**GOAL: HbA1c below 7% or as low as possible without significant hypoglycemia.**

**Algorithm:**



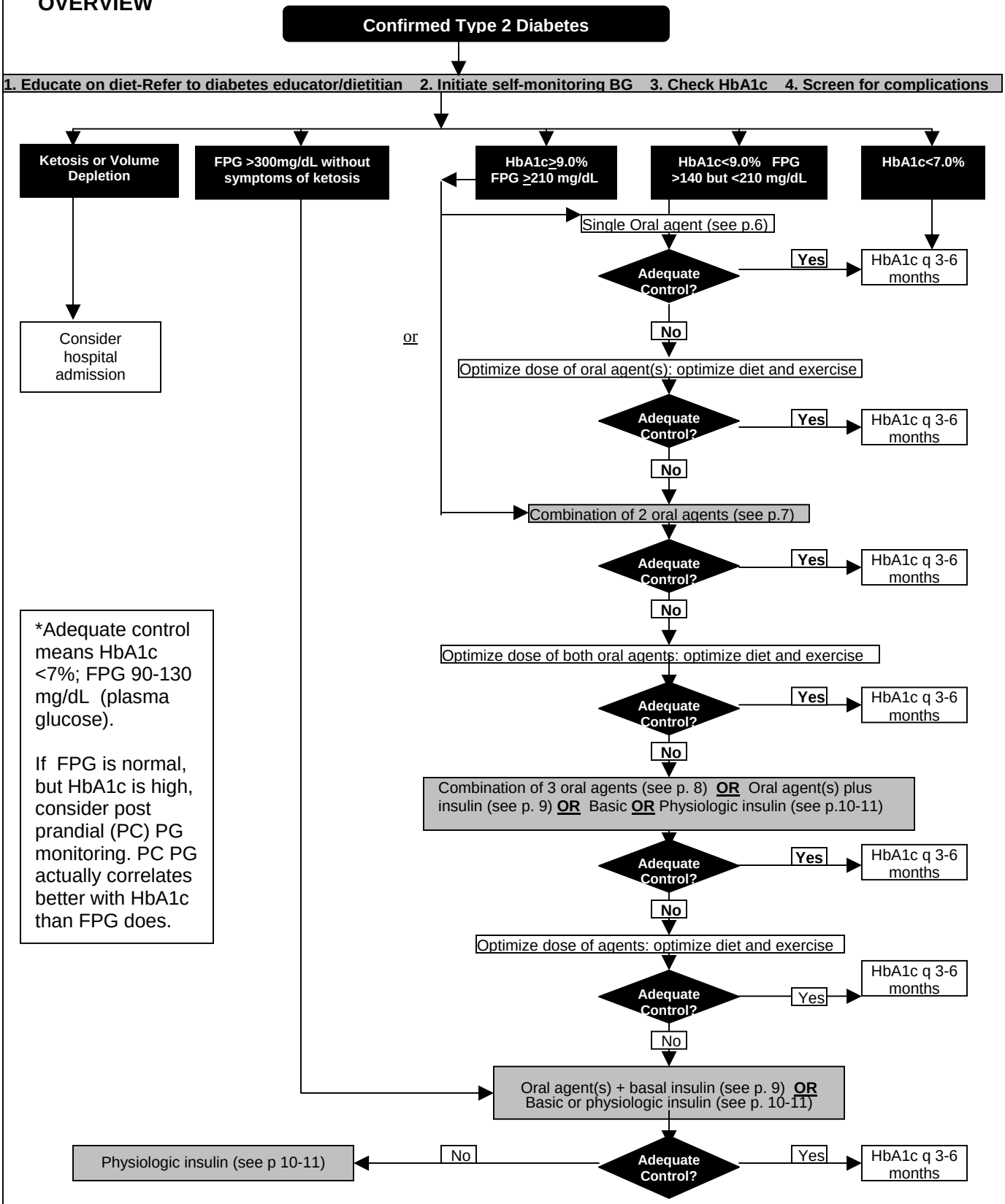

**Note:** Occasionally HbA1c values do not accurately reflect glycemic control. If serum glucose levels are higher than would be predicted by an HbA1c, consider measuring serum fructosamine.

The table to the right provides an approximate comparison of *average* plasma glucose (PG) and HbA1c values

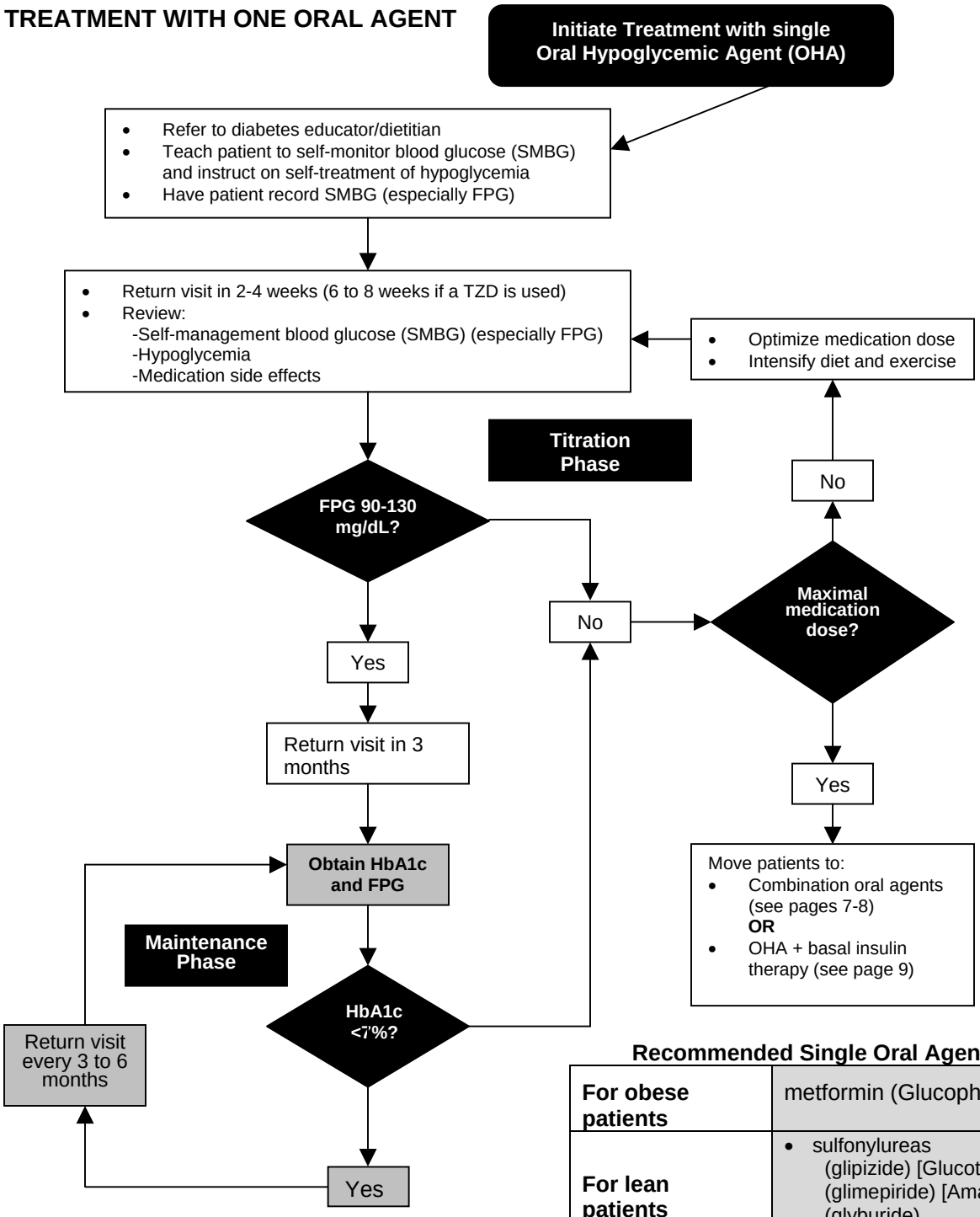
| Plasma Glucose | HbA1c % |
|---|---|
| 345 | 12 |
| 310 | 11 |
| 275 | 10 |
| 240 | 9 |
| 205 | 8 |
| 170 | 7 |
| 135 | 6 |

# MEDICAL MANAGEMENT

## OVERVIEW



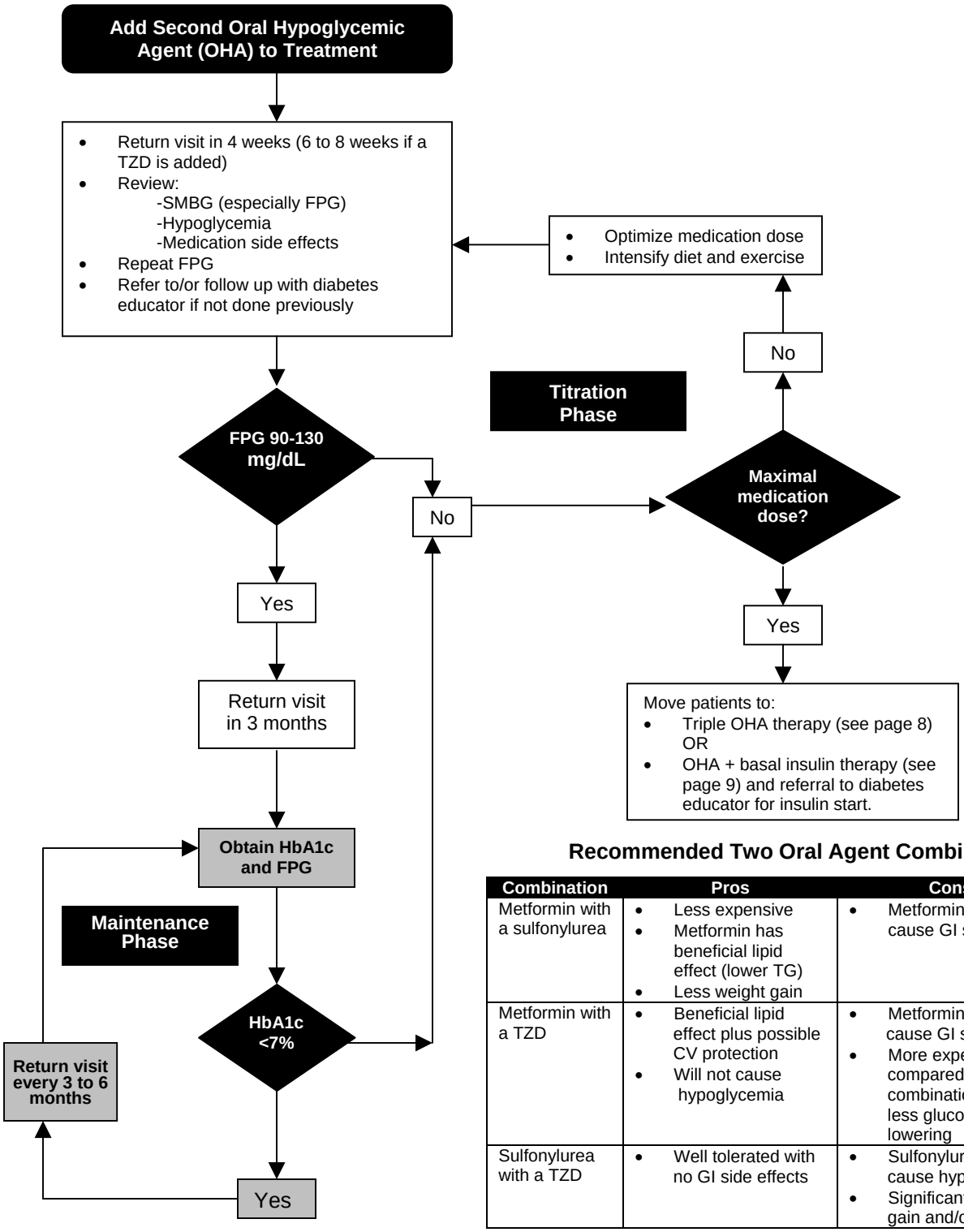
## TREATMENT WITH ONE ORAL AGENT




**Recommended Single Oral Agents**

| | |
|---|---|
| **For obese patients** | metformin (Glucophage) |
| **For lean patients** | • sulfonylureas (glipizide) [Glucotrol] (glimepiride) [Amaryl] (glyburide)<br>OR<br>• metformin |
| **Other Choices** | • rosiglitazone (Avandia)<br>• pioglitazone (Actos) |

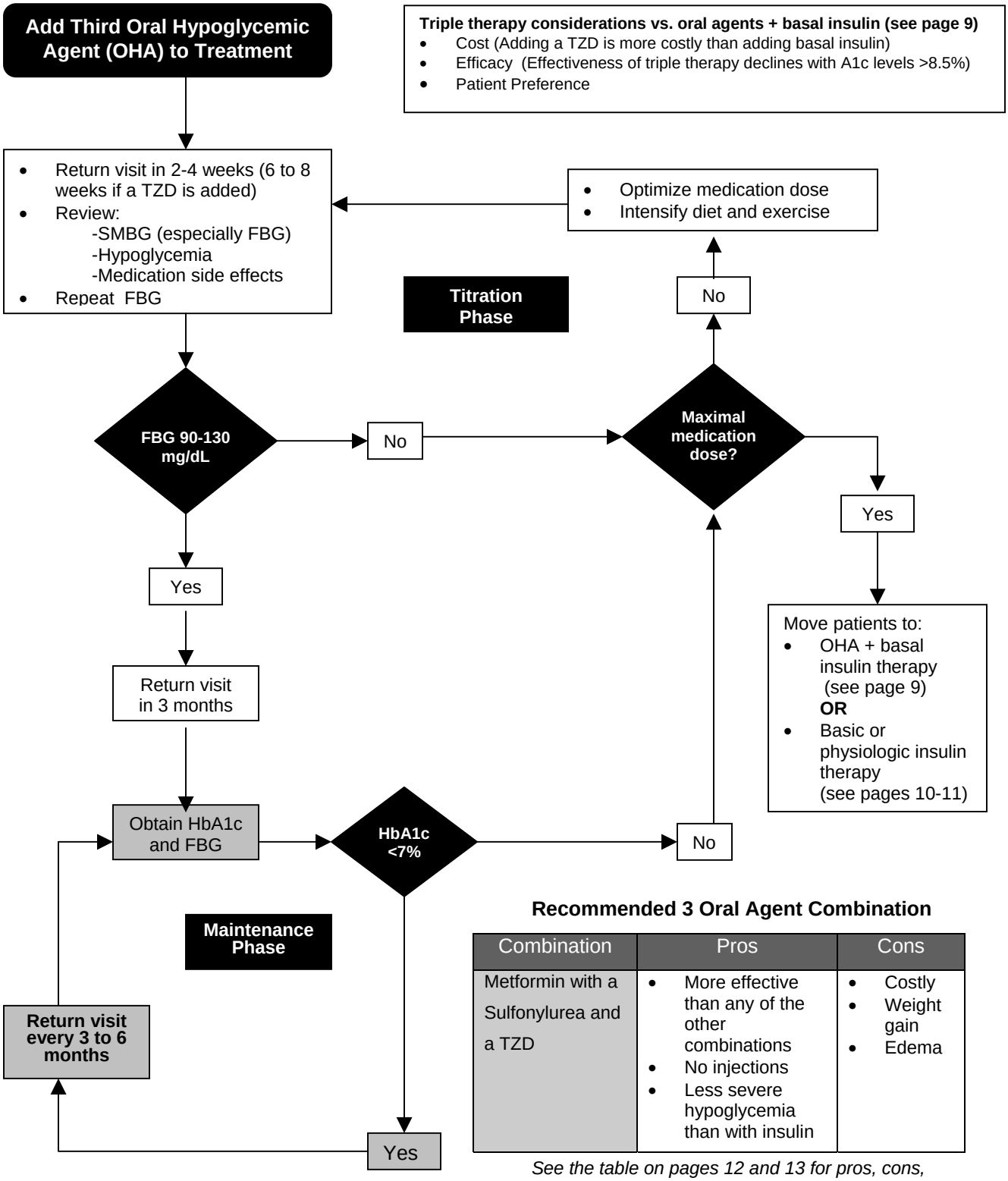
## TREATMENT WITH 2 ORAL AGENTS




### Recommended Two Oral Agent Combinations

| Combination | Pros | Cons |
|---|---|---|
| Metformin with a sulfonylurea | • Less expensive<br>• Metformin has beneficial lipid effect (lower TG)<br>• Less weight gain | • Metformin may cause GI side effects |
| Metformin with a TZD | • Beneficial lipid effect plus possible CV protection<br>• Will not cause hypoglycemia | • Metformin may cause GI side effects<br>• More expensive compared to other combinations, with less glucose lowering |
| Sulfonylurea with a TZD | • Well tolerated with no GI side effects | • Sulfonylureas may cause hypoglycemia<br>• Significant weight gain and/or edema |

## TREATMENT WITH 3 ORAL AGENTS

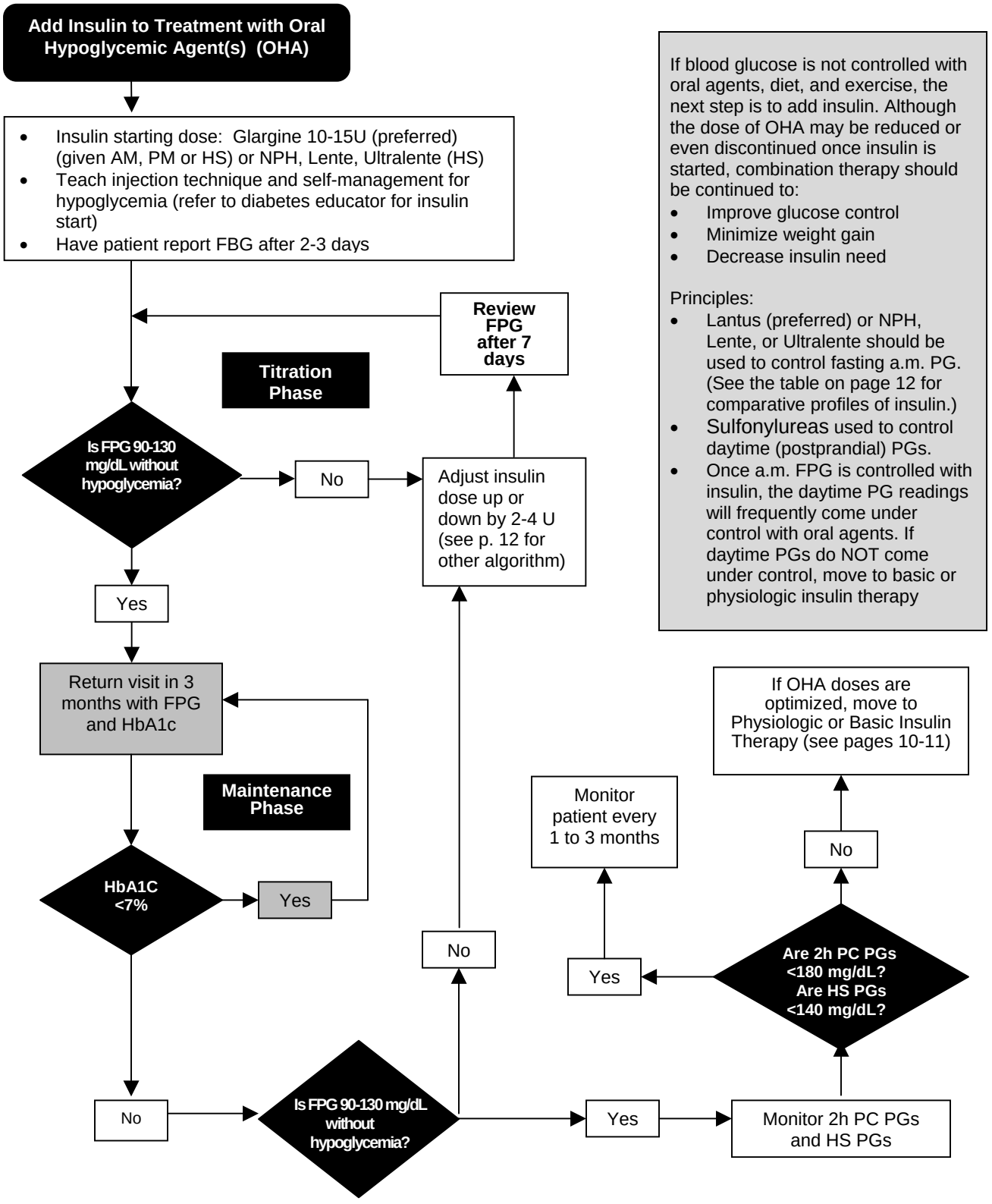



**Recommended 3 Oral Agent Combination**

| Combination | Pros | Cons |
|---|---|---|
| Metformin with a Sulfonylurea and a TZD | • More effective than any of the other combinations<br>• No injections<br>• Less severe hypoglycemia than with insulin | • Costly<br>• Weight gain<br>• Edema |

*See the table on pages 12 and 13 for pros, cons, dosing and cost for oral agents*

## ORAL AGENT(S) PLUS INSULIN




If blood glucose is not controlled with oral agents, diet, and exercise, the next step is to add insulin. Although the dose of OHA may be reduced or even discontinued once insulin is started, combination therapy should be continued to:

- Improve glucose control
- Minimize weight gain
- Decrease insulin need

Principles:

- Lantus (preferred) or NPH, Lente, or Ultralente should be used to control fasting a.m. PG. (See the table on page 12 for comparative profiles of insulin.)
- Sulfonylureas used to control daytime (postprandial) PGs.
- Once a.m. FPG is controlled with insulin, the daytime PG readings will frequently come under control with oral agents. If daytime PGs do NOT come under control, move to basic or physiologic insulin therapy

## BASIC INSULIN THERAPY

### Principles

- Basic insulin regimens are NOT designed to mimic normal insulin physiology.
- Basic insulin regimens are NOT recommended for type 1 patients.
- Basic insulin regimens are sometimes adequate for control of type 2 patients who have failed maximum efforts with oral medications or oral medications plus insulin.
- Basic insulin regimens are sometimes chosen when patients are not able to involve themselves in a physiologic multiple daily dose regimen.
- Consistency with meals and adequate adherence to a medical nutrition therapy plan are important to success of basic insulin regimens.
- Patients on basic insulin therapy regimens should move to physiologic (basal/bolus) insulin if goals are not met with basic insulin therapy.

### Sample Basic Insulin Regimens

The following are some basic insulin regimens. See the table on page 12 for comparative profiles of insulin.

### Pre-mixed Insulins

All of the following are BID (pre-breakfast and pre-supper)

- 70/30 (NPH Regular)
- 70/30 (NPA NovoLog)
- 75/25 (NPL Humalog)

### Split-mixed Insulins

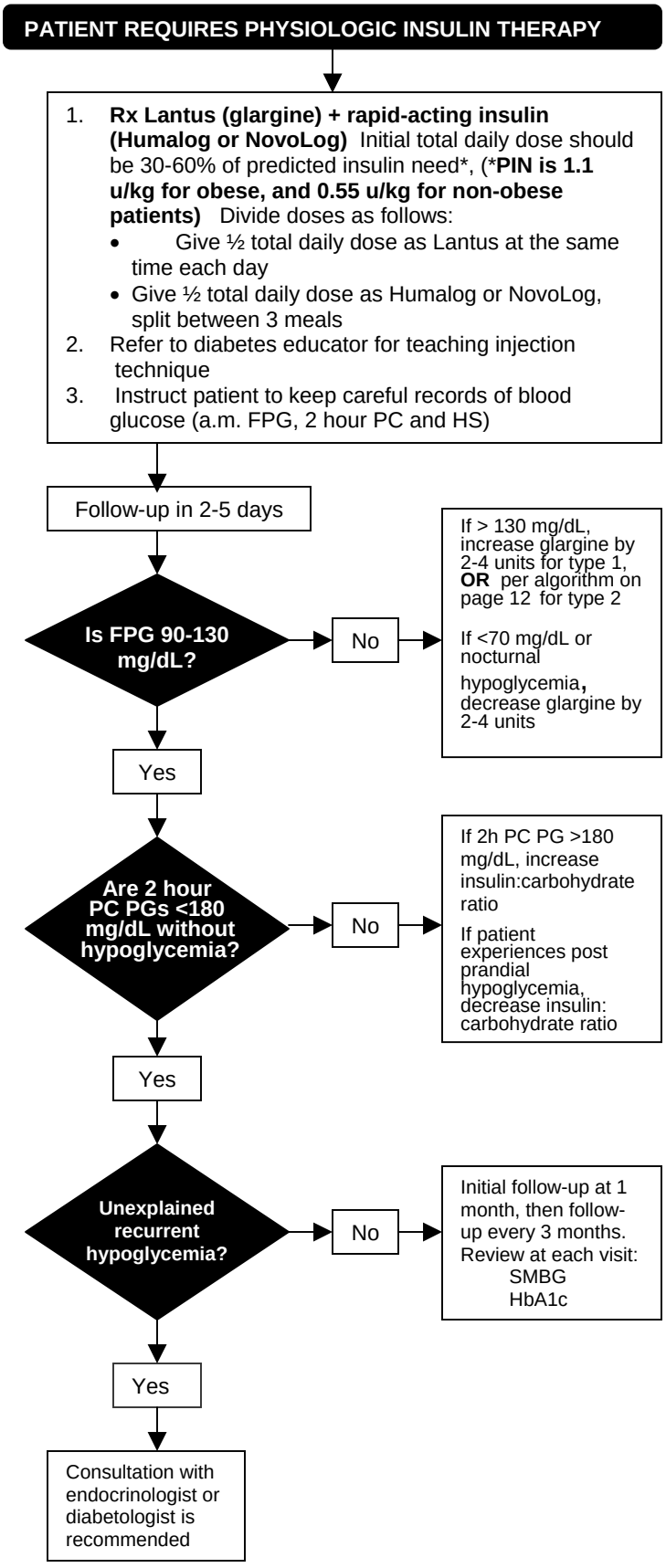
- NPH or Lente BID (either a.m. and supper, or a.m. and HS) with:
  - Regular insulin before breakfast and before supper
    OR
  - Humalog or NovoLog before breakfast and before supper

## PHYSIOLOGIC (BASAL/BOLUS) INSULIN

### Principles

- This more intensive insulin regimen provides closest approximation to normal insulin physiology. It uses Lantus insulin for basal metabolic control, and Humalog or NovoLog for prandial control and correction of high glucose levels.
  - Lantus is used to control glycemia in the basal state when not eating. The period from bedtime until breakfast is the best reflection of this. Bedtime snacking is NOT recommended.
  - Rapid-acting insulin (Humalog or NovoLog) is added prior to each meal (See the algorithm on the following page for recommended initial dosing). This insulin is adjusted to prevent postprandial hyperglycemia or hypoglycemia. Late postprandial blood sugar (4 hours after a meal) should be equal to pre-meal blood sugar.
  - Pre-meal Humalog or NovoLog doses are usually determined by carbohydrate counting and use of a carbohydrate ratio. A less commonly used strategy is pre-meal insulin based on a fixed meal plan. **In either case, training in medical nutrition therapy by a qualified dietitian, and training in insulin use by a qualified diabetes educator, are recommended for success.**
- Almost all type 1 patients should be on basal/bolus regimens. Most type 2 patients requiring insulin will benefit from basal/bolus insulin to attain good control.
- Instruction for modifying insulin doses for exercise and sick days should be incorporated into the regimen.

## INITIAL PHYSIOLOGIC INSULIN REGIMEN




### Using the 1500 Rule

The 1500 rule can be used to guide the patient's dosage of insulin in two circumstances:

- To calculate a sliding scale to determine a correction dose for a high PG reading
- To calculate insulin-to-carbohydrate ratio (i.e., to anticipate insulin needed to cover the carbohydrate content of a meal)

**To calculate a sliding scale:**

1. **Determine the current total daily dose (TDD):** Add up ALL the insulin the patient takes in a 24 hour period (short + long-acting).
2. **Divide 1500 by the TDD.** This is the predicted amount (mg/dL) the PG will lower for every 1 unit of Humalog or NovoLog insulin added.
3. **Increase Humalog or NovoLog** by the number of units needed to reduce the PG to an appropriate level (<140 mg/dL)).
4. **Encourage the patient to keep careful records** of resulting PG readings. (Most helpful readings are a.m. FPG, 2-3 hours PC, and HS).

   *Example*
   1. Patient takes 50 units of insulin/day **(TDD = 50**)
   2. **1500/50 = 30** (which means 1 unit of insulin will lower PG by 30 points)
   3. So, if PG is 170, use 1 extra unit to drop it to 140. If PG is 200, use 2 extra units, and so on

**To calculate insulin-to-carb ratio:**

1. **Determine the current total daily dose (TDD):** Add up ALL the insulin the patient takes in a 24-hour period (short + long-acting)
2. **Divide 1500 by the TDD.** This is the predicted amount (mg/dL) the BG will lower for every 1 unit of Humalog or NovoLog insulin added.
3. **Multiply predicted PG lowering (mg/dL) x .33** This is the number of grams of carbohydrate covered by 1 unit of insulin. (For most people, a starting dose would be 1 unit of Humalog or NovoLog insulin for every 10-15 grams of carbohydrate to be eaten.)

   *Example*
   1. Patient takes 50 units of insulin/day **(TDD = 50)**
   2. **1500/50 = 30** (which means 1 unit of insulin will lower BG by 30 points)
   3. **30 X .333 = 10** (which means patient will need 1 unit of insulin for every 10 grams of carbohydrate anticipated in a meal)

# MEDICATIONS

### GLARGINE (Lantus) or NPH TITRATION ALGORITHM (as basal insulin)

Start with 10-15 units/day and adjust weekly

| Mean of FPG values from preceding 2 days | Increase insulin dose/day |
|---|---|
| >180 mg/Dl | 8 units |
| 140-180 mg/dL | 6 units |
| 120-139 mg/dL | 4 units |
| 100-119 mg/dL | 2 units |

Riddle MC et al: *Diabetes Care* (2003) 26: 3080-86

### COMPARATIVE PROFILES OF INSULIN

| Insulin | Description | Onset | Peak | Usual Effective Duration | Usual Maximum Duration | January 2004 AWP |
|---|---|---|---|---|---|---|
| **Humalog/NovoLog** | Clear | 15 min | 1-1½ hrs | 1-3 hrs | 3-4 hrs | 10 ml: $67 |
| Regular | Clear | 30-60 min | 2-3 hrs | 3-6 hrs | 4-8 hrs | 10 ml: $31 |
| NPH | Cloudy | 2-4 hrs | 4-10 hrs | 10-16 hrs | 14-18 hrs | 10 ml: $31 |
| Lente | Cloudy | 3-4 hrs | 4-12 hrs | 12-18 hrs | 16-20 hrs | 10 ml: $31 |
| Ultralente | Cloudy | 6-10 hrs | unknown | 18-20 hrs | 20-30 hrs | 10 ml: $31 |
| Glargine (Lantus )* | Clear | 1 hr | none | 24 hrs | 24 hrs | 10 ml: $51 |

* Notes about Lantus:

- Lantus is preferred over other long-acting insulin because of better control of a.m. fasting PG with decreased hypoglycemia, especially nocturnally. Must be given at same time each day.
- Due to improved stability of PG control, Lantus appears to be the optimum insulin for combination with oral agents or as basal insulin in physiologic insulin therapy.
- Administer Lantus once daily for individuals with type 1 and type 2 who require basal (long-acting) insulin for control of hyperglycemia—in a.m. with oral agents, and at supper or HS in physiologic insulin therapy in combination with short-acting insulin.
- Lantus cannot be diluted or mixed with other types of insulin or solutions.
- Administer Lantus subcutaneously ONLY—not to be given IV.

### ORAL AGENT MEDICATION SUMMARY

| | Generic Name | Brand Name | Dosing | 30 Day AWP January 2004 | Pros | Cons |
|---|---|---|---|---|---|---|
| **Metformin** | metformin | Glucophage | 500 mg BID (start) to 1000 mg BID | **Generic:**<br>500 mg BID $42<br>850 mg BID $72<br>1000 mg BID $87 | ▪ Prevents weight gain (preferred for obese patients-most type 2 diabetics)<br>▪ Favorable lipid effects<br>▪ No hypoglycemia<br>▪ Maximum PG effect at 3-4 weeks<br>▪ Decreases formation of advanced glycosylation end products (AGE)<br>▪ Most benefit derived at 1500-1700 mg/day | ▪ GI distress (nausea/diarrhea)<br>▪ CAUTION - increased risk of acidosis: **- STOP MED with acute illness, dehydration, or IV contrast dyes**<br>▪ DO NOT use for patients with chronic liver disease, CHF, renal failure (e.g., creatinine<br>▪ $\geq$1.5 men or $\geq$ 1.4 women), alcoholism, or decreased creatinine clearance in elderly |
| | metformin XR | Glucophage XR | 500-1500 mg @ supper | **Generic:**<br>500 mg QD $23<br>1500 mg QD $67<br>2000 mg QD $89 | | |
| **Sulfonylureas** | glipizide XL | Glucotrol XL | 5 mg QD to 20 mg QD (max) [may give dose BID] | **Generic:**<br>5 mg QD $13<br>10 mg QD $25 | ▪ Preferred for lean patients (small % of type 2)<br>▪ Well tolerated<br>▪ Can be combined with oral agents except Prandin, Starlix, or other sulfonylureas<br>▪ Maximum PG effect at 5-7 days<br>▪ Most benefit derived at 50% of maximum dose | ▪ Higher risk for hypoglycemia and weight gain<br>▪ Do not use with Prandin or Starlix, or other sulfonylureas |
| | glimepiride | Amaryl | 1 mg QD to 8 mg QD (max) | **Brand Only:**<br>1 mg QD $12<br>2 mg QD $19<br>4 mg QD $37 | | |

## ORAL AGENT MEDICATION SUMMARY (continued)

| | Generic Name | Brand Name | Dosing | 30 Day AWP January 2004 | Pros | Cons |
|---|---|---|---|---|---|---|
| Combination | metformin + glipizide | Metaglip | 2.5 mg/500 mg QD to BID<br><br>5 mg/500 mg QD to BID | **Brand Only:**<br>2.5 mg/500 mg BID $64<br><br>5 mg/500 mg BID $64 | ▪ Less expensive<br>▪ Metformin has beneficial lipid effects (lower triglycerides)<br>▪ Less weight gain | ▪ Metformin may cause GI side effects<br>▪ Do not use with Prandin or Starlix |
| | metformin + glyburide | Glucovance | 1.25 mg/250 mg BID w/meals<br><br>5 mg/500 mg BID to TID w/meals | **Brand Only:**<br>1.25 mg/250 mg BID $59<br><br>5 mg/500 mg BID $69 | | |
| | metformin + Avandia (rosiglitazone) | Avandamet | 2 mg/500 mg 1-2 BID<br><br>4 mg/500 mg 1-2 BID | **Brand Only:**<br>2 mg/500 mg 1 BID $106<br><br>4 mg/500 mg 1 BID $172 | ▪ Beneficial lipid effects plus possible CV protection<br>▪ Does not cause hypoglycemia | ▪ Metformin may cause GI side effects<br>▪ More expensive when compared to other combinations, with less glucose lowering |
| TZDs | pioglitazone | Actos | 15-45 mg QD (increasing dose slowly may decrease edema) | **Brand Only:**<br>15 mg QD $109<br>30 mg QD $175<br>45 mg QD $189 | ▪ Good option for patients who are intolerant of metformin<br>▪ Does not cause hypoglycemia<br>▪ Lowers serum insulin<br>▪ Favorable lipid effects (best with Actos); may help further decrease TG levels<br>▪ Appears to have prolonged benefits with lower secondary failure rate<br>▪ Improves many CV risk factors and may be cardiovascular protective | ▪ Edema can be significant, especially if given with insulin<br>▪ Fluid retention may lead to or exacerbate heart failure (**in this circumstance, drug should be stopped)**<br>▪ Consider checking LFTs q 2 mos x 1 yr; but appears risks are low or absent<br>▪ Expensive<br>▪ Actos may change metabolism of birth control pills<br>▪ Slow onset; maximum effect takes 6-12 weeks |
| | rosiglitazone | Avandia | 2-8 mg QD (increasing dose slowly may decrease edema) | **Brand Only:**<br>2 mg QD $64<br>4 mg QD $91<br>8 mg QD $168 | | |
| Miglitinides | repaglinide | Prandin | 0.5 mg PC with each meal<br><br>16 mg/day (max) | **Brand Only**:<br>0.5mg TID $99<br>1mg TID $99<br>2mg TID $99 | ▪ Short-acting; increases insulin release PC<br>▪ Lower risk of hypoglycemia than sulfonylureas (similar mechanism of action) | ▪ Frequent dosing<br>▪ Increased cost<br>▪ Do not use with sulfonylureas<br>▪ Does not control FBG as well as sulfonylureas |
| | nateglinide | Starlix | 120 mg PC TID (start and maintenance)<br><br>60 mg PP TID if HbA1c is close to goal | **Brand only:**<br>120 mg TID $112<br><br>60 mg TID $108 | ▪ Improves first-phase insulin release<br>▪ Can be used as monotherapy or in combination with other oral agents (except sulfonylureas)<br>▪ May lower risk of hypoglycemia | ▪ Frequent dosing<br>▪ Increased cost<br>▪ Do not use with sulfonylureas<br>▪ Does not control FBG as well as well as sulfonylureas |
| A lpha Glucosdiase Inhibitors | acarbose | Precose | 25 mg TID (start) to 100 mg TID (max) | **Brand Only:**<br>25 mg TID $65<br>50 mg TID $70<br>100 mg TID $83 | ▪ Does not cause hypoglycemia<br>▪ Not a systemic agent<br>▪ Inhibits/delays digestion of ingested carbohydrates | ▪ Increased cost<br>▪ GI side effects common<br>▪ Frequent dosing<br>▪ Modest impact on HbA1c |
| | miglitol | Glyset | 50-100 mg TID with meals | **Brand Only:**<br>50 mg TID $74<br>100 mg TID $87 | | |

# MANAGEMENT OF RELATED CONDITIONS

## CARDIOVASCULAR DISEASE

Patients with diabetes have 2- to 4-fold increased risk of coronary heart disease (CHD). The risk for CHD is increased much more dramatically in women with diabetes. All individuals with diabetes have a higher fatality rate once they have CHD than those without diabetes.

### Multifactorial Intervention to Reduce Risk

**Interventions that are well established**

Research has established that modification of certain risk factors commonly associated with diabetes can substantially reduce the risk of cardiovascular disease. Well established interventions are listed below. Persons with diabetes benefit from these interventions to an extent that exceeds that seen in non-diabetic patients.

- Antiplatelet therapy
- Smoking cessation
- LDL cholesterol lowering
- Control of blood pressure

**Interventions with supporting risk reduction data**

Other issues with some data supporting a role in reduction of cardiovascular risk including the following:

- Administration of ACE inhibitors (MICRO-HOPE study)
- Treatment of diabetic dyslipidemia (This term refers to elevated triglyceride and low HDL cholesterol. Most often these two conditions are seen together, but some patients will have isolated low HDL cholesterol. These lipid abnormalities have been identified as secondary targets of therapy in diabetes after correction of LDL cholesterol elevations.)

**Emerging risk factors**

Emerging risk factors are those factors for which there appears to be substantial association with cardiovascular risk, but for which evidence is currently lacking to show that modification reduces risk. Consensus of opinion on how to handle these emerging risk factors (listed below) is still evolving.

- High sensitivity C-reactive protein (hsCRP)
- Homocysteine levels

### CV Screening

Patients with symptoms suggesting CHD should undergo evaluation using exercise stress testing, myocardial perfusion imaging, stress echocardiography, or coronary angiography.

The value of screening of the asymptomatic individual for CHD is still of some uncertainty. While CHD is much more prevalent among those with diabetes, there is little evidence that screening procedures in asymptomatic persons has a positive effect on outcomes. As of yet, there is no consensus of opinion on which diagnostic test is the best choice for evaluating the asymptomatic patient. Nevertheless, consider screening by exercise stress test (with or without perfusion imaging) based on accumulated risk factors, the presence of other vascular disease, or abnormalities on a resting EKG.

## CARDIOVASCULAR DISEASE (continued)

### CV Medication Recommendations

**Beta blockers**

Patients with known coronary artery disease (CAD), especially if they have had a coronary event, may benefit from beta blockers.

**Aspirin Therapy**

- *Secondary prevention:* Use aspirin therapy as a *secondary* prevention strategy in men and women with diabetes who already have evidence of large-vessel disease. This includes diabetic individuals with a history of myocardial infarction, vascular bypass procedure, stroke or transient ischemic attack, peripheral vascular disease, claudication, and/or angina
- *Primary prevention:* Use aspirin therapy as a *primary* prevention strategy for high-risk men and women who have type 1or type 2 diabetes. This includes individuals with diabetes who have one or more of the following:
  - Family history of CHD
  - Cigarette smoking
  - Hypertension
  - Overweight (Body Mass Index [BMI] $\geq$25)
  - Albuminuria (micro or macro)
  - Hyperlipidemia:
    - Tchol >200 mg/dL
    - LDL >100 mg/dL
    - HDL <45 mg/dL for men<br>HDL <55 mg/dL for women
    - TG >150 mg/dL
  - Age >40 yrs
- *Individuals who may NOT be candidates for aspirin therapy:*
  - Individuals with diabetes under the age of 40 without CV risk factors listed above
  - People with aspirin allergy, bleeding tendency, anticoagulation therapy, recent gastrointestinal bleeding, and clinically active hepatic disease

**ASPIRIN DOSAGE RECOMMENDATION:**
**Enteric-coated aspirin in doses of 81-325 mg/day**

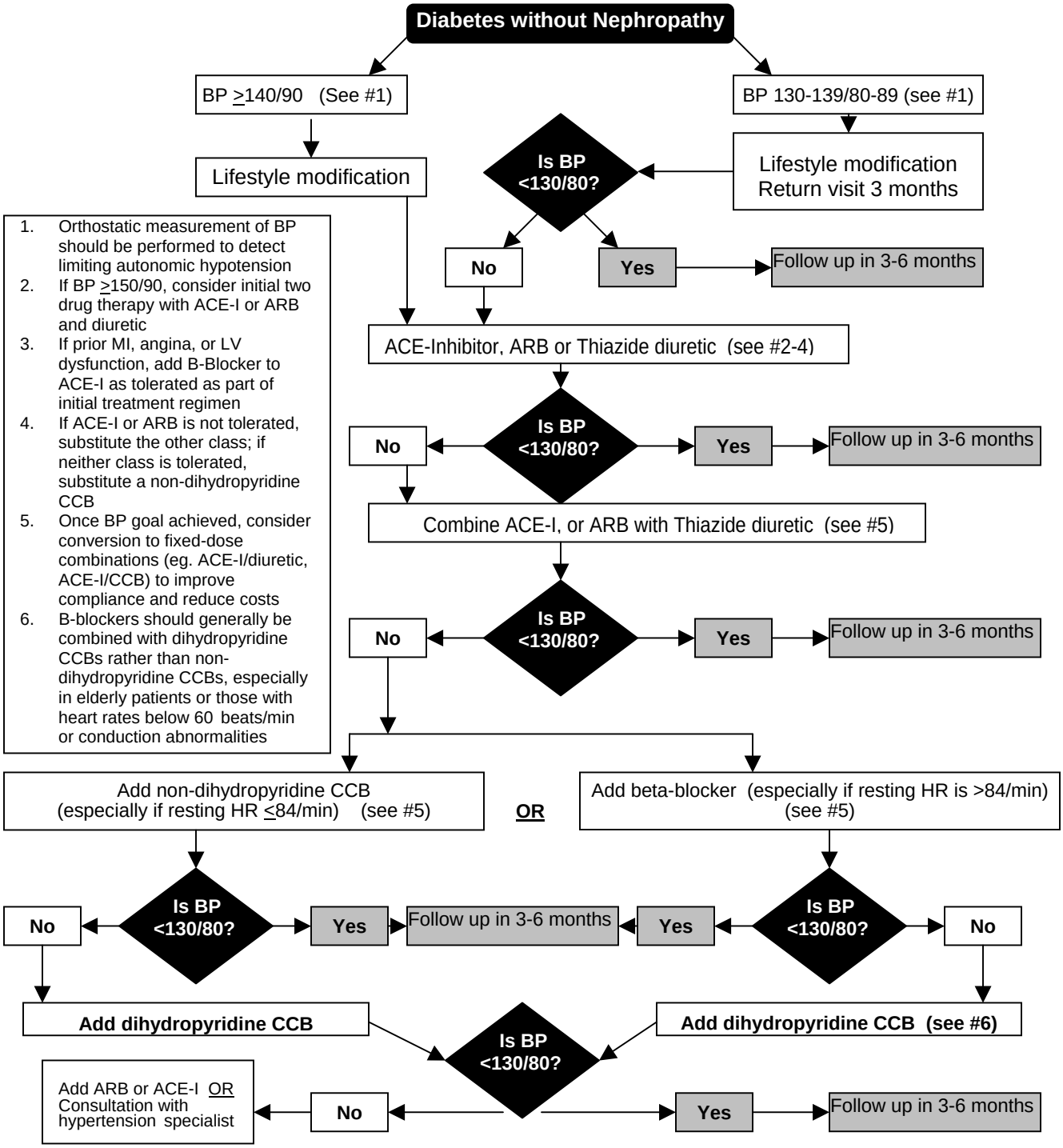
## BLOOD PRESSURE CONTROL WITHOUT NEPHROPATHY

Aggressive treatment of high blood pressure in people with diabetes can reduce their cardiovascular risk. Lowering blood pressure to <130/80 mm Hg has a beneficial effect in reducing diabetic complications that is at least equal to the effect of glucose control. Microvascular complications of diabetes have also been shown to occur less frequently with lower blood pressure

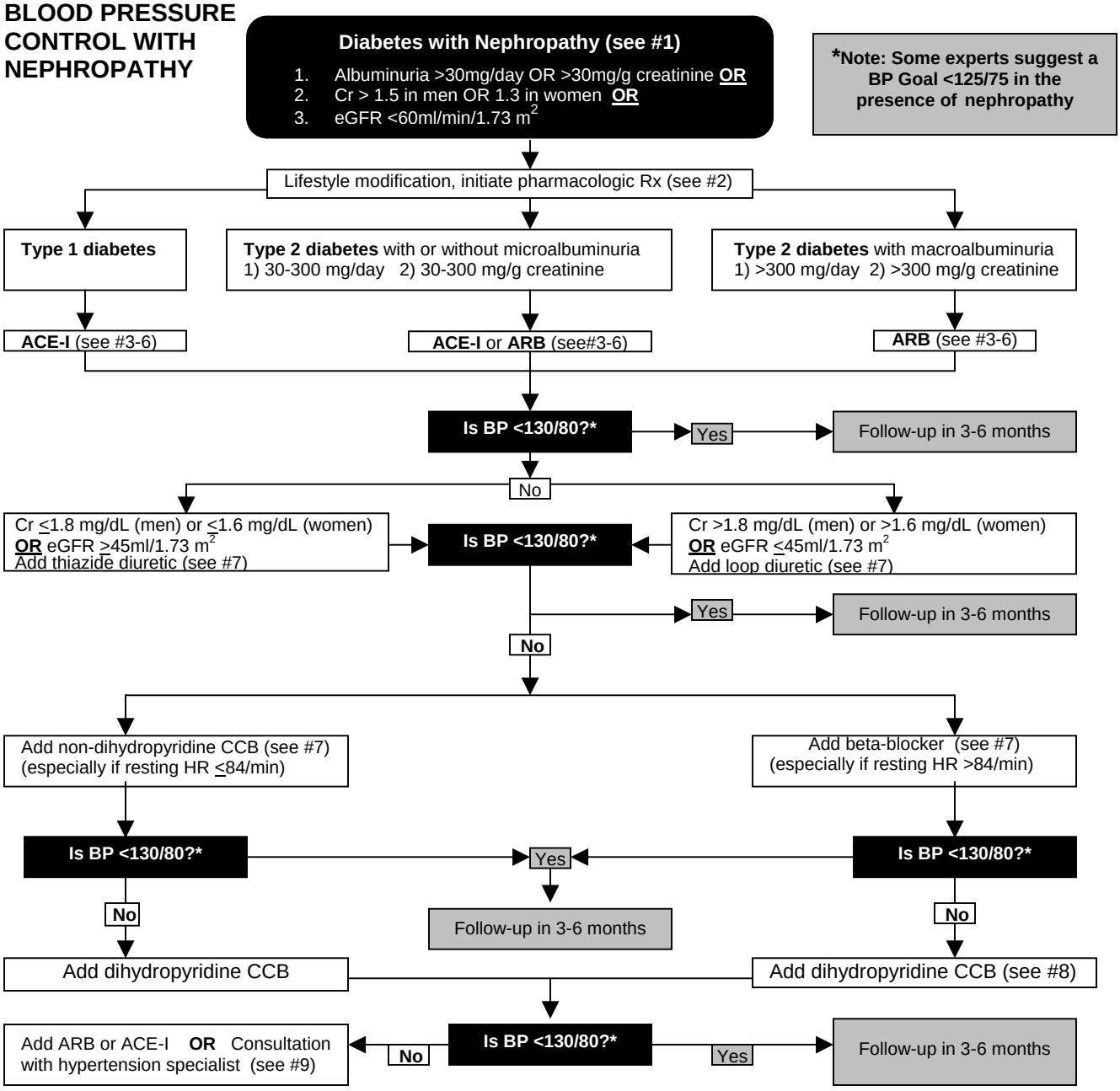
**Goals:** (CHECK AT EACH OFFICE VISIT)

- **<130/80 mm Hg for patients without nephropathy**
- **Some experts would lower the BP goal to <125/75 in the presence of nephropathy (see page 17)**

**Note:** The majority of patients require more than one medication to control blood pressure to these levels**.**



## BLOOD PRESSURE CONTROL WITH NEPHROPATHY




1. Orthostatic measurement of BP should be performed to detect limiting automonic neuropathy with orthostatic hypotension
2. The finding of albuminuria in type 1 or 2 diabetic patients is an indication for treatment with an ACE-I or ARB irrespective of BP levels
3. If BP ≥150/90, consider initial two drug therapy with ACE-I or ARB and diuretic
4. If prior MI, angina, or LV dysfunction, add beta-blocker to ACE-I as tolerated as part of initial treatment
5. If ACE-I or ARB is not tolerated, substitute the other class; if neither class is tolerated, substitute a non-dihydropyridine CCB
6. Renal function and serum potassium must be closely monitored in patients with renal insufficiency. ACE-I and ARBs are relatively contraindicated in patients with Cr≥3.0 or eGFR <20 ml/min. If renal function deteriorates significantly (>25-35%), with ACE-I or ARB, consider work-up for renal artery disease
7. Once BP goal is achieved, consider conversion to fixed-dose combinations (e.g. ACE-I/diuretic, ACE-I/CCB) to improve compliance and reduce costs
8. Beta-blockers should generally be combined with dihydropyridine CCBs rather than non-dihydropyridine CCBs, especially in elderly patients or those with heart rates below 60 beats/min or conduction abnormalities. Combination therapy can cause severe bradycardia and cardiac syncope
9. Combining ACE-I and ARB may lower BP and reduce proteinuria; long-term studies are not available

## HYPERTENSION MEDICATION RECOMMENDATIONS

### Cardiovascular disease protection in hypertension

- **ACE Inhibitors**
  The HOPE and EUROPA trials have suggested that ACE inhibitors may be cardioprotective in patients at high risk for cardiovascular disease, including patients with diabetes; the patients in these trials did not all have hypertension. In contrast, in the ALLHAT Hypertension Trial, the thiazide-like diuretic, chlorthalidone, was at least as effective as the ACE inhibitor lisinopril, in preventing cardiovascular complications in hypertensive patients with diabetes mellitus.

- **ARBS**
  ARBS have not yet been documented to be more cardioprotective than other drugs in hypertensive patients with diabetes mellitus. In the LIFE Trial they did provide superior cardiovascular protection to a beta-blocker-based regimen in patients with and without diabetes.

### Renal disease protection in hypertension

- ACE inhibitors have specific renal protective effects in type 1 patients with diabetic nephopathy.
- ARBs have specific renal protective effects in type 2 patients with diabetic nephropathy.
- Both ACE inhibitors and ARBs slow progression from microalbuminuria to macroalbuminuria in type 2 patients with diabetes

### Current recommendations:

- For patients with no albuminuria**,** the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7) recommends an ACE inhibitor, an ARB, or a thiazide diuretic for initial therapy in patients with hypertension and diabetes. The ADA recommends that all patients with diabetes and hypertension be treated with a regimen that includes an ACE inhibitor or an ARB.
- For patients with type 1diabetes and any degree of albuminuria, the ADA recommends ACE inhibitors based on clinical trial support. For patients with type 2 diabetes and micro-albuminuria, either ACE inhibitors or ARBs are reasonable initial choices. ARBs should be strongly considered for type 2 diabetes patients with hypertension and macroalbuminuria (>300 mg/day).
- The efficacy of combining ACE inhibitors with ARBs – either for lowering blood pressure, reducing proteinuria, or preventing renal complications – has not yet been definitively demonstrated in clinical trials

### Diuretics

Thiazide diurectics benefit patients with diabetes and hypertension, either as initial therapy in patients without albuminuria or as part of a combined regimen.
Thiazide diuretics are minimally effective in patients with estimated GFR below about 45 ml/1.73m$^2$, which correlates with a serum creatinine of about 1.8 mg/dL in men and 1.6 mg/dL in women.
Loop diuretics may be necessary in these patients. Short-acting loop diuretics (furosemide, bumetanide) may need to be given bid-tid for hypertension control.

### Beta-Blockers

Beta-blockers have demonstrated benefits in treating hypertension in persons with diabetes especially as part of a multi-drug regimen. However, beta-blockers may be less effective than ARBs for preventing cardiovascular complications in diabetes (LIFE Trial). Adding beta- blockers to ACE inhibitors may not reduce blood pressure as effectively as adding diuretics or calcium channel blockers to ACE inhibitors, especially in patients with heart rates below 84 beats/minute. Beta-blockers should be part of the treatment regimen for most patients with diabetes who are post-myocardial infarction or who have left ventricular dysfunction or angina. Their adverse metabolic effects – weight gain, elevation of blood glucose and triglycerides, and reduction of HDL cholesterol—are not absolute contraindications to their use.

### Calcium Channel Blockers (CCBs)

Calcium channel blockers are useful components of combination therapy to reduce blood pressure in persons with diabetes. Long-acting dihydropyridine and non-dihydropyridine CCBs have both been shown to reduce cardiovascular complications in patients with diabetes as compared to placebo. CCBs prevent cardiovascular complications as effectively as ACE inhibitor and diuretics in all categories except heart failure, where they are significantly inferior. Direct comparisons of dihydropyridine CCBs and non-dihydropyridine CCBs are not available with respect to cardiovascular or renal complications. However, non-dihydropyridine CCBs may be preferred in patients with diabetes and proteinuria, although dihydropyridine CCBs may also be safely used in these patients as long as there is concurrent therapy with an ACE inhibitors or ARB. Beta-blockers should generally be combined with dyhydropyridine CCBs rather than non-dihydropyridine CCBs, especially in elderly patients or patients with conduction abnormalities or baseline heart rates below 60 beats/minute. Combining the two classes of CCBs may effectively lower blood pressure in some patients with difficult to control hypertension.

### Alpha-blockers

The ALLHAT study raises questions about potential adverse effects of the alpha-blocker doxazosin on incidence of cardiovascular outcomes. Until this has been further studied, alpha blockers should not be an initial choice for add-on therapy.

American Diabetes Association. Hypertension management in adults with diabetes. Diabetes Care 2004; (Suppl 1): S65-67

National High Blood Pressure Education Program Coordinating Committee. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003: 42:1206-1252

## HYPERLIPIDEMIA

People with type 2 diabetes have an **increased prevalence of lipid abnormalities.** Therapy aimed at improving lipids has been shown to reduce macrovascular disease and mortality in these patients. Glycemic control can also beneficially lower plasma lipids, especially triglyceride levels (TG), and **probably reduce cardiovascular risk.**

Clinical trials have shown that diabetic patients with CHD benefit more than other CHD patients from lipid-lowering therapy. The American Diabetes Association has concluded that the primary emphasis should be placed on lowering LDL levels, but interventions to lower TG and raise HDL levels may also be useful. Data suggest treatment with statins may be appropriate for people >40 with total cholesterol ≥135 mg/dL.

### Lipid Therapy Goals

**Optimal LDL levels** **<70-100 mg/dL**
In patients with vascular disease the LDL should be less than 70.

**Optimal HDL levels** **Men >40 mg/dL**
**Women >50 mg/dL**

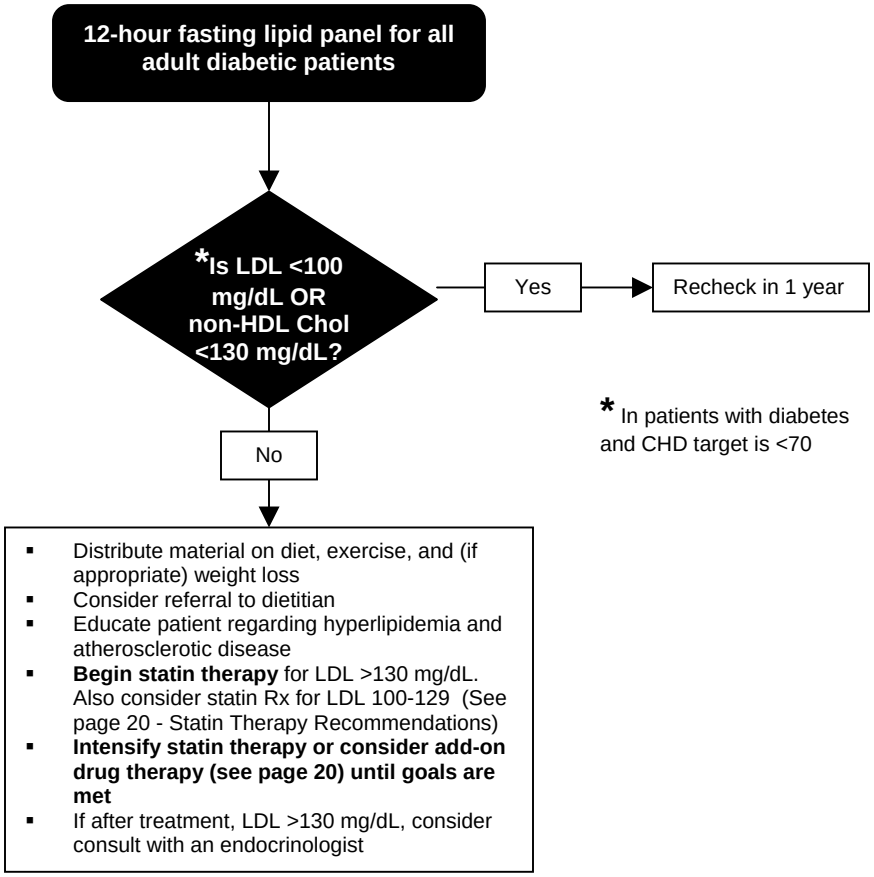
**Desirable TG levels** **<150 mg/dL**

Patients with TGs >1000 mg/dL demand immediate attention to get this level below 400 mg/dL.

**Note:** For patients with high triglycerides, some authorities have recently discussed an alternate goal for treatment as **non-HDL cholesterol <130 mg/dL.** Non-HDL cholesterol is defined as total cholesterol minus HDL cholesterol.

### Algorithm

Pharmacological therapy is indicated if there is an inadequate response to lifestyle modifications and glycemic control. Statins are usually the drugs of choice.



## STATIN THERAPY RECOMMENDATIONS

1. **Optimize statin therapy first, THEN add-on therapy options indicated in paragraph 2 below if needed**

2. **If patient is still unable to attain treatment goal with optimized statin therapy, consider the following options based on the patient's situation:**

   - **If LDL level remains > above the individual's target,** consider add-on therapy with Zetia.[1]

   - **If TG remain > 400 mg/dL,** a careful history of the patient's alcohol consumption should be obtained. Factors that may increase TG are listed to the right.

| Factors that may increase triglycerides |
|---|
| Alcohol intake |
| Excessive carbohydrate intake |
| Poor glycemic control (improve HbA1c <7) |
| Oral estrogen therapy (discontinue estrogen or convert to transdermal estrogen) |
| Most beta blockers (discontinue beta blocker or convert to carvedilol) |

   - **If TG remain > 400 mg/dL,** and/or HDL <40 mg/dL (even in the rare patient with LDL <100 mg/dL), despite maximized statin, obtain good glycemic control and consider add-on therapy with a fibric acid derivative or niacin.[2] This combination is not well studied. It **requires patient consent and careful monitoring** and is associated with an increased risk for myositis or rhabdomyolysis.

   - **If goals remain unmet,** consider referral to an endocrinologist or lipid specialist.

**Notes:**

[1] Zetia should be used in conjunction with a statin only when lipid goals cannot be met with a statin alone. Zetia should NOT be used as a single agent unless the patient cannot tolerate a statin. There are no outcome data for the use of Zetia.

[2] If niacin is used, it should be done so with caution in patients with peptic ulcer disease or gout. Use of niacin in most patients does not cause a significant deterioration of glucose control but occasionally patients may experience worsening of glucose control.

## NEUROPATHY

Foot problems, including acquired structural deformation, ulceration, and wound-healing failure, are frequent causes of morbidity and mortality in people with diabetes. Ulceration and wound-healing failure are frequent causes for lower extremity amputation. Once the amputation of one limb has occurred, the prognosis for the contralateral limb is poor.

Loss of sensation (neuropathy) may be the first sign leading to acquired deformity and/or amputation. Patients who can feel a monofilament line applied with 10 grams of pressure on selected sites most likely will not develop foot ulcers or acquired deformities. **Thus, the emphasis should be on identifying diabetic patients with high-risk feet, specifically feet with loss of protective sensation or with significant vascular disease.**

The foot evaluation should include careful questioning about claudication, evaluation of pulses, inspection of the feet, and a monofilament fiber examination. Consider a non-invasive vascular exam for patients without palpable pulses.

All identified high-risk patients should undergo a comprehensive program of patient education, including instruction on daily self-care and guidelines on appropriate footwear. Sometimes prescription footwear is helpful. Medicare will provide yearly reimbursement for the following items for diabetic patients with high-risk feet:

- One pair of extra-depth shoes and three pairs of inserts, or
- One custom-molded shoe plus two additional pairs of inserts

Diabetic foot ulcers require extensive evaluation. The evaluation should include the following:

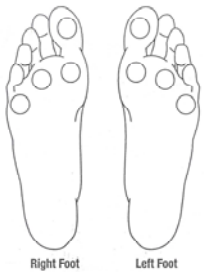
- Assessment of the surrounding tissue for signs of edema, cellulitis, and/or abscess.
- Evaluation for exudate, necrosis, and sinus tracts (The ability to gently probe through an ulcer to bone has been shown to be highly predictive of osteomyelitis)
- Evaluation of circulation to involved extremity

Diabetic foot ulcers are often polymicrobial: the primary cause is pressure. The goal for treatment must be removal of the pressure from the involved area. Early specialty consultation is encouraged.

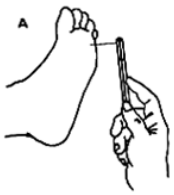
### Monofilament Application Instructions

The sensory testing device used with the diabetic foot exam is a nylon filament mounted on a holder that has been standardized to deliver a 10-gram force when properly applied.

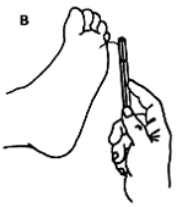
**The sites to be tested are indicated below:**




1. Apply the monofilament perpendicular to the skin's surface as shown below. The approach, skin contact, and departure of the filament should be approximately 1 ½ seconds in duration. Each site should be tested at least twice and use of sham testing is recommended. Apply the filament along the perimeter of, NOT on, an ulcer site, callus, scar, or necrotic tissue.




2. Apply sufficient force to cause the filament to bow into a C-shape as show below. Do not allow the filament to slide across the skin or to make repetitive contact at the test site.




3. Randomize the selection of test sites and time between successive tests to reduce the potential for patient guessing.
4. Ask the patient to respond "yes" when the filament is felt. Record the response

**Monofilaments available through:**

Lower Extremity Amputation Prevention Program
1-888-275—4772 (Press 1- HRSA information)
One time 50 monofilament order at no charge

Medical Monofilament Manufacturing
(508) 746-7877 (Disposable unit @ $0.30 each)

Reusable units @ $10 each can be ordered by calling: (800) 543-9055; or (225) 923-1297

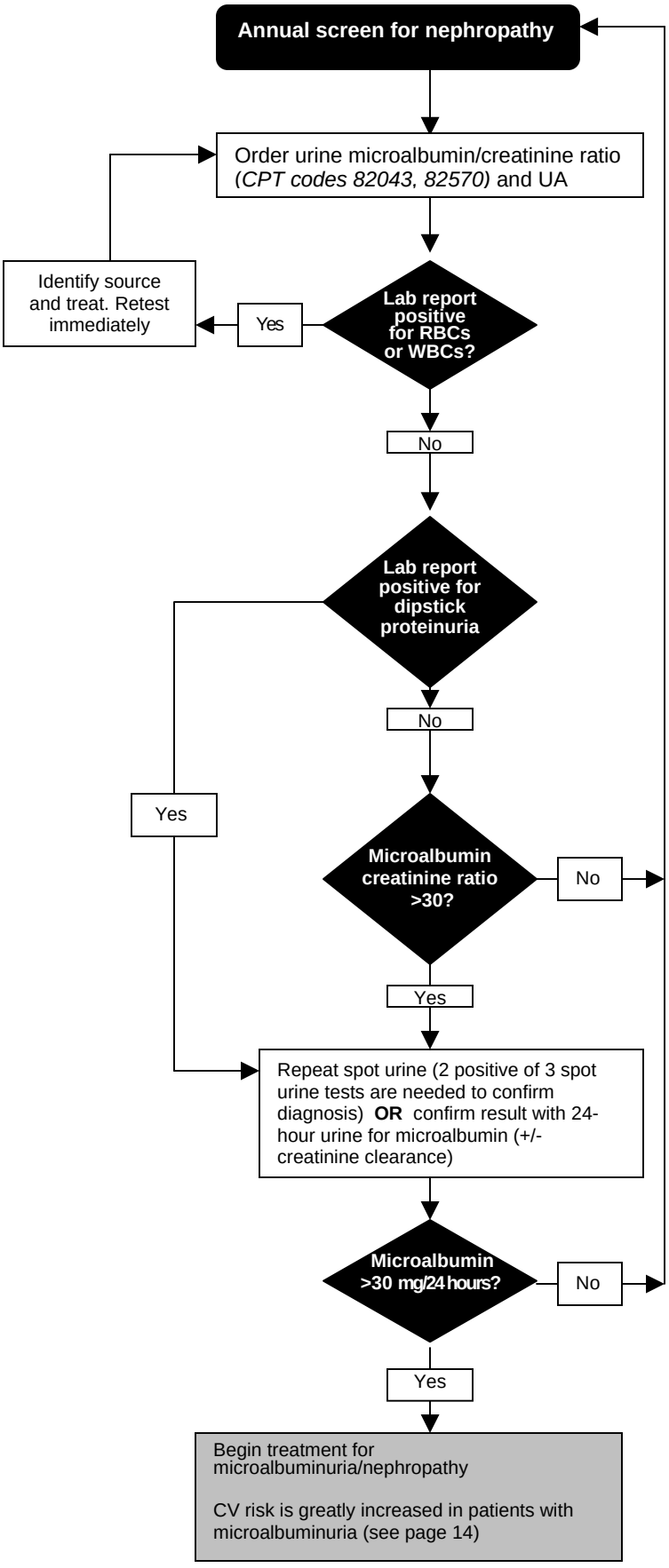
## NEPHROPATHY

The onset of diabetic kidney disease can be detected at the earliest stage by testing for increased albumin excretion in the urine.

**Normal:** <30 mg/24 hours or <30 mg/gram of creatinine

The risk of progression of early diabetic kidney disease can be markedly reduced by the following:

- Maintenance of good glucose control (HbA1c <7.0%)
- Use of ACE inhibitors or ARBs even in normotensive subjects
- Blood pressure control ≤130/80 (Some experts suggest <125/75 for patients with nephropathy)
- Dietary protein restriction (0.8 g/kg/day initially and 0.6 g/k/day if creatinine clearance starts to fall)



## RETINOPATHY AND DIABETIC EYE DISEASE

Diabetes is the leading cause of blindness in the United States for adults 20-74. Many of the early signs of diabetic retinopathy (notable on a dilated fundus examination) are asymptomatic for the patient. Early treatment can be the key to prevention of blindness.

The American Diabetes Association recommends an initial, and thereafter an annual, dilated and comprehensive eye exam by an ophthalmologist or optometrist who is knowledgeable and experienced in the diagnosis and management of diabetic retinopathy. Less frequent exams (2-year intervals) may be considered with the advice of an eye care professional for individual patients in good control and a normal exam. Patients with diagnosed diabetic retinopathy and patients with diabetes with prior normal eye exams who are, or become, pregnant should be referred promptly to an ophthalmologist.

Recommended Eye Examination Schedule for Type 1 and Type 2 Diabetes

| Type of Patient | Minimum Routine Follow-up |
|---|---|
| **Type 1** patients should have a dilated eye exam by an optometrist or ophthalmologist three to five years after diagnosis (Some evidence suggests that microvascular complications may develop before age 10 among those diagnosed as infants and toddlers)<br><br>**Type 2** patients should have a dilated eye exam immediately following diagnosis for diabetes or pre-diabetes | **Annually for most patients** with mild or no non-proliferative diabetic retinopathy (NPDR) or microaneurysms<br><br>**Biennially for patients in good control**, prior normal exam and with advice of an eye care professional<br><br>**More frequent examination** is required with moderate or progressive mild NPDR |
| **Pregnancy:** women should have a dilated eye exam when planning pregnancy if possible, and also during the first trimester (Does not apply to women with gestational diabetes since they are not at increased risk for diabetic retinopathy) | **First trimester,** with continued close follow-up. Diabetic patients who become pregnant may experience accelerated diabetic retinopathy and should be monitored closely by an ophthalmologist |
| **Patients with any macular edema, severe non-proliferative diabetic retinopathy (NPDR) or any proliferative diabetic retinopathy (PDR)** | **Refer promptly** to an ophthalmologist experienced in the treatment of diabetic retinopathy* |
| **Patients with vision loss** from diabetes should be encouraged to pursue visual rehabilitation | **Refer** to an ophthalmologist or an optometrist who is trained or experienced in low-vision care |

* Do not delay referral to an ophthalmologist until PDR develops. Early referral is very important for patients with type 2 diabetes and severe NPDR, since laser treatment at this stage is associated with a 50% reduction in risk of severe visual loss and vitrectomy.

**Source:** ADA Clinical Practice Recommendations, 2004. *Diabetes Care* 27 (Suppl. 1): S86

## CERTIFIED DIABETES EDUCATION PROGRAMS

Diabetes self-management training (DSMT) is generally conducted in a hospital or clinic with group and individual instruction. DSMT consists of education from a 'team' of individuals from various disciplines. The 'team' may include nurses, dietitians, doctors, pharmacists, exercise physiologists, health educators, counselors and other knowledgeable health care professionals**.** An *individualized* program is based on an initial assessment, and may cover any or all of these topics depending on the needs of the patient:

- The diabetes disease process and treatment options
- Incorporating physical activity into a lifestyle
- Monitoring blood glucose, urine ketones (when appropriate), and using results to improve control
- Preventing, detecting and treating acute complications
- Goal setting to promote health, and solve problems of daily living
- Incorporating appropriate nutritional management
- Utilizing medications for therapeutic effectiveness
- Integrating psychosocial adjustment to daily life
- Promoting preconception care, management of pregnancy, and gestational diabetes
- Preventing (through risk reducing behavior), detecting, and treating chronic complications

**The Utah Diabetes Prevention and Control Program (DPCP) and the American Diabetes Association (ADA) provide certification necessary for insurance reimbursement by most health insurance plans. Note that Medicare reimburses only for diabetes education provided in ADA recognized programs.**

### Box Elder, Cache, Davis, and Weber Counties

| | | |
|---|---|---|
| **Brigham City Hospital** | | |
| Brigham City, Utah 84302 | 435-734-4339 | DPCP |
| **Bear River Valley Hospital** | | |
| Tremonton, Utah 84337 | 435-257-7441 | ADA |
| **Budge Clinic** | | |
| Logan, Utah 84341 | 435-792-1707 | ADA |
| **Logan Regional Hospital** | | |
| Logan, Utah 84341 | 435-716-5439 | ADA |
| **Bountiful Health Center** (IHC) | | |
| Bountiful, Utah 84010 | 801-294-1000 | ADA |
| **Lakeview Hospital** | | |
| Bountiful, Utah 84010 | 801-299-2470 | ADA |
| **Endocrine and Diabetes Clinic** (McKay-Dee) | | |
| Ogden, Utah 84403 | 801-387-7900 | ADA |
| **McKay Dee Outpatient Diabetes Education** | | |
| Ogden, Utah 84403 | 801-387-7520 | ADA |

### Salt Lake County

| | | |
|---|---|---|
| **Alta View Hospital** | | |
| Sandy, Utah 84070 | 801-314-2894 | ADA |
| **Bryner Clinic** | | |
| Salt Lake City, Utah 84102 | 801-519-7165 | ADA |
| **Cottonwood Hospital** | | |
| Murray, Utah 84106 | 801-314-2894 | ADA |
| **Cottonwood Family Practice** | | |
| Salt Lake City, Utah 84121 | 801-262-3443 | ADA |
| **Cottonwood Internal Medicine** | | |
| Murray, Utah 84107 | 801-314-4300 | ADA |
| **Diabetes Specialty Center** | | |
| Salt Lake City, Utah 84106 | 801-483-1100 | DPCP |
| **Granger Medical Clinic Pharmacy** | | |
| West Valley City, Utah 84120 | 801-965-3639 | DPCP |
| **Holladay Health Clinic** (IHC) | | |
| Salt Lake City, Utah 84124 | 801-408-1840 | ADA |
| **Jordan Valley Hospital** | | |
| West Jordan, Utah 84088 | 801-562-4263 | ADA |
| **LDS Hospital** | | |
| Salt Lake City, Utah 84143 | 801-314-2899 | ADA |
| **Medical Tower Family Practice** | | |
| Murray, Utah 84107 | 801-314-4266 | ADA |
| **Medical Tower Specialty Clinic** | | |
| Murray, Utah 8407 | 801-314-4890 | ADA |
| **Memorial Medical Center** | | |
| Salt Lake City, Utah 84105 | 801-461-7979 | ADA |
| **Primary Children's Outpatient Education** | | |
| Salt Lake City, Utah 84113 | 801-588-2711 | DPCP |
| **Sandy Health Center (IHC)** | | |
| Salt Lake City, Utah 84094 | 801-501-2100 | ADA |
| **St. Marks Hospital** | | |
| Salt Lake City, Utah 84124 | 801-268-7358 | ADA |
| **Salt Lake Clinic** | | |
| Salt Lake City, Utah 84102 | 801-535-8117 | ADA |
| **Taylorsville Health Center** (IHC) | | |
| Salt Lake City, Utah 84118 | 801-840-2100 | ADA |
| **Utah Diabetes Center, University Medical Center** | | |
| Salt Lake City, Utah 84108 | 801-581-7761 | ADA |
| **West Jordan Health Center** (IHC) | | |
| West Jordan, Utah 84088 | 801-256-6343 | ADA |

## CERTIFIED DIABETES EDUCATION PROGRAMS (continued)

### Central and Southwestern Utah

| | | |
|---|---|---|
| **Central Valley Medical Center** | | |
| Nephi, Utah 84648 | 435-623-3092 | ADA |
| **Garfield Memorial Hospital** | | |
| Panguitch, Utah 84759 | 435-676-8842 | ADA |
| **Gunnison Valley Hospital** | | |
| Gunnison, Utah 84634 | 435-528-3955 | DPCP |
| **Dixie Regional Medical Center** | | |
| St. George, Utah 84770 | 435-688-5085 | ADA |
| **River Road Clinic** (IHC) | | |
| St. George, Utah 84770 | 435-688-6200 | ADA |
| **Sevier Valley Hospital** | | |
| Richfield, Utah 84701 | 435-893-0371 | DPCP |
| **Valley View Medical Center** | | |
| Cedar City, Utah 84720 | 435-868-5000 | ADA |

### Uintah Basin and Southeastern Utah

| | | |
|---|---|---|
| **Allen Memorial Hospital** | | |
| Moab, Utah 84532 | 435-259-7191 | DPCP |
| **Ashley Valley Medical Center** | | |
| Vernal, Utah 84078 | 435-789-3342 X 174 | ADA |
| **Castleview Hospital** | | |
| Price, Utah 84501 | 435-636-4822 | DPCP |
| **Monument Valley Health Center** | | |
| Monument Valley, Utah 84536 | 435-727-3242 | DPCP |
| **Utah Navajo Health Systems, Inc.** | **435-651-3291** | |
| Blanding Family Practice Clinic | | DPCP |
| Montezuma Creek Clinic | | DPCP |
| Navajo Mountain Clinic | | DPCP |
| **Uintah Basin Medical Center** | | |
| Roosevelt, Utah 84066 | 435-722-6131 X 1363 | ADA |

### Utah and Wasatch Counties

| | | |
|---|---|---|
| **American Fork Hospital** | | |
| American Fork, Utah 84003 | 801-763-3471 | ADA |
| **Mountain View Hospital** | | |
| Payson, Utah 84651 | 801-465-7045 | ADA |
| **Heber Valley Medical Center** | | |
| Heber City, Utah 84032 | 435-654-2500 | ADA |
| **Utah Valley Regional Medical Center** | | |
| Provo, Utah 84605 | 801-357-7546 | ADA |

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